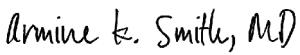


Exhibit 13

Specific Causation Expert Report for Allan Howard Armine K Smith, MD

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		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition	Chart 4 Deposition/FM
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
TCE	5,937	153,943	668,552	660,782	1,019,982
PCE	251	6,472	28,107	27,780	42,882
VC	343	8,859	38,473	38,026	58,697
BZ	70	1,831	7,952	7,859	12,132

10. Causation Analysis

I have reviewed the general causation expert reports of Drs. Hatten and Bird. The analysis in those reports supports my opinions in this report and finds that the four chemicals in the water at Camp Lejeune are causally related to kidney cancer. I also researched and read the epidemiology, toxicology and mechanistic evidence that exists relating to the toxins at issue in this case and agree that the toxins, as they existed in combination in the water at Camp Lejeune, are causally related to kidney cancer under a more likely than not standard, which exceeds the “at least as likely as not” standard in this case. A summary of some of the evidence is below and is used for purposes of weighing the potential harmful effects of the exposure Mr. Howard had to the water at Camp Lejeune.

a. TCE and Kidney Cancer

Trichloroethylene (TCE) is a widely used industrial solvent and volatile organic compound (VOC) known to contaminate soil and groundwater, as seen at Camp Lejeune. It has been classified as a *known human carcinogen* by both the International Agency for Research on Cancer (IARC) and the Environmental Protection Agency (EPA), with specific evidence linking TCE exposure to kidney cancer.²

i. Epidemiological Evidence

IARC concludes there is sufficient evidence in humans for TCE's carcinogenicity, particularly causing kidney cancer.² The Agency for Toxic Substances and Disease Registry (ATSDR) also recognizes sufficient evidence of causation for kidney cancer associated with TCE.¹ A 2010 meta-analysis by Kelsh et al. demonstrated a statistically significant relative risk (RR) of 1.42 (95% CI 1.17-1.77) for occupational TCE exposure and kidney cancer.³ A 2011 EPA manuscript reported an overall RR of 1.27 (95% CI 1.13-1.43), with higher risks for groups exposed to elevated TCE levels (RR 1.58, 95% CI 1.28-1.96).⁴

ii. Meta-Analyses and Studies

Karami et al. (2012) reviewed 9 cohort studies and found an elevated RR of 1.26 (95% CI 1.02-1.56) for TCE exposure and renal cancer, with consistent results across cohort and case-control designs.⁵ The study noted that misclassification of exposure in earlier research likely underestimated the true risk.⁵

iii. Mechanistic Evidence

TCE is metabolized into nephrotoxic compounds, such as S-(1,2-dichlorovinyl)-L-cysteine (DCVC), which bioactivate in the kidneys, causing DNA damage and mutations. TCE exposure induces oxidative stress, leading to lipid peroxidation and impaired antioxidant activity, which are key drivers of renal carcinogenesis.

iv. Conclusion

In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on all uses of trichloroethylene (TCE) to safeguard public health from the associated risks, including kidney cancer, linked to TCE exposure.⁶

Mr. Howard's levels of TCE are significantly higher than the levels in the literature that show a causal association between TCE and RCC. According to Bove 2014a's cumulative exposure charts, Mr. Howard would have been categorized as being in the "medium" exposure group for TCE.⁷ This was associated with a 1.21 HR.⁷ Mr. Howard meets and exceeds other levels in the literature causally associating TCE with kidney cancer.

Collectively, epidemiological data, mechanistic studies, and meta-analyses provide robust evidence of the causal link between TCE exposure and kidney cancer, highlighting its significant public health implications. It is overwhelmingly probable that TCE causes kidney cancer.

b. PCE and Kidney Cancer

Perchloroethylene (PCE), also known as tetrachloroethylene, is a volatile organic compound widely used in the dry-cleaning industry and as a degreaser. It is classified as a *probable human carcinogen* by IARC (Group 2A),² and the EPA has determined that PCE is "Likely to be Carcinogenic to Humans" by all exposure routes.⁸

i. Epidemiological Evidence

Cape Cod Study: Aschengrau et al. examined individuals exposed to PCE-contaminated drinking water, finding a relative risk (RR) of 1.23 (95% CI 0.40-3.11) for kidney cancer with any exposure.⁹ Although underpowered, this study was very similar to the circumstances at Camp Lejeune, including the levels of exposure, and therefore provides very relevant information. Aschengrau also found a relative risk (RR) of 1.36 for low exposures relating to kidney cancer.⁹

U.S. Kidney Cancer Study: Purdue et al. conducted a case-control study and reported an odds ratio (OR) of 3.1 (95% CI 1.3-7.4) for high cumulative PCE exposure and kidney cancer, indicating a strong association.¹⁰

Dry-Cleaning Industry Studies: Ruder et al. found a standardized mortality ratio (SMR) of 1.41 (95% CI 0.46-3.30) among 1,708 dry-cleaning workers exposed to PCE.¹¹ Karami et al. found an elevated risk of developing renal cell carcinoma (OR) of 2.0 (95% CI: 0.9-4.4), which increased for longer employment in the dry-cleaning industry to 2.5 (95% CI 0.4-14.4).¹² Callahan et al. identified a dose-response relationship, with the highest exposure group showing a hazard ratio (HR) of 13.2 (95% CI 1.9-90.8) for kidney cancer mortality.¹³

ii. Camp Lejeune Study

Bove et al. examined civilian workers at Camp Lejeune and found a standardized mortality ratio (SMR) of 1.30 (95% CI 0.52-2.67) for kidney cancer.¹⁴ In Bove 2014a, with particular respect to PCE, the relative risks of kidney cancer based on low, medium and high exposures were: 1.40 for low exposure, 1.82 for medium exposure and 1.59 for high exposure.⁷ Mr. Howard would meet the medium exposure group threshold with a RR of 1.82.

iii. Mechanistic Evidence

Metabolic Activation: PCE is metabolized into trichloroacetic acid (TCA) and other metabolites that can form DNA adducts, leading to mutations and carcinogenesis.

Oxidative Stress: PCE exposure induces reactive oxygen species (ROS) and lipid peroxidation, which cause DNA strand breaks and mutations.

Cytotoxicity and Proliferation: PCE and its metabolites trigger cytotoxic effects, promoting compensatory cell proliferation, which increases cancer risk.

iv. Conclusion

In December 2024, the U.S. Environmental Protection Agency (EPA) implemented a ban on perchloroethylene (PCE) to protect public health from the associated risks, including its link to various cancers including kidney cancer at low levels.⁶

Mr. Howard's levels of PCE are significantly higher than the levels in the literature that show a causal association between PCE and RCC. According to Bove 2014a's cumulative exposure charts,

Mr. Howard would have been categorized as being in the “Medium” exposure group for PCE.⁷ This corresponded to a HR of 1.82.⁷ Mr. Howard meets additional levels of PCE exposure shown in the literature to be associated with kidney cancer.

Epidemiological studies, particularly those involving occupational and environmental exposures, along with mechanistic data, support a causal association between PCE and kidney cancer. The findings underscore the carcinogenic potential of PCE. It is more likely than not that PCE causes kidney cancer.

c. Other Contaminants: Vinyl Chloride and Benzene

Both compounds are linked to genotoxic effects but are less robustly associated with RCC than TCE. Synergistic effects with other carcinogens (e.g., TCE) amplify the risk.

i. Epidemiological Evidence

Benzene has a significant body of literature that shows a causal relationship with kidney cancer. A meta-analysis was performed analyzing 29 studies and found that there was a relative risk (RR) of 1.2 (95% CI 1.03 – 1.39).¹⁵ Additionally, Hu (2002) found a monotonic response relationship between benzene and kidney cancer (RCC) and a RR of 1.8 (95% CI: 1.2-2.6) for kidney cancer in men exposed to benzene.¹⁶ There are other studies as well showing this causal relationship, including the Camp Lejeune studies.^{7,14}

Vinyl Chloride has been less studied than the other three chemicals at issue. However, when it has been studied there has been epidemiologic literature showing a causal association between vinyl chloride and kidney cancer. For example, the Hu (2002) study found a monotonic response relationship between vinyl chloride and kidney cancer in men with an OR of 2.0 (95% CI: 1.2 – 3.3).¹⁶ Further, Bove et al. 2014a supports this causal relationship.⁷

ii. Mechanistic Evidence

Benzene’s carcinogenicity in the kidney is driven by its metabolism into reactive intermediates, including hydroquinone and benzoquinones, which generate oxidative stress and DNA damage. These metabolites induce DNA strand breaks, chromosomal aberrations, and mutations, contributing to genomic instability. Benzene exposure also impairs DNA repair mechanisms and forms DNA adducts, further exacerbating mutagenesis. These pathways collectively increase the risk of renal carcinogenesis.

Vinyl chloride is metabolized into reactive intermediates like chloroethylene oxide and chloroacetaldehyde, which form DNA adducts, such as etheno-deoxyadenosine. These adducts disrupt genomic integrity and lead to mutations in key genes regulating DNA repair and cell cycle control. Additionally, vinyl chloride induces oxidative stress and TP53 pathway dysregulation, both of which are implicated in RCC development. Its toxic effects on renal cells underscore its role in kidney carcinogenesis.

iii. Conclusion

Mr. Howard's levels of Benzene and VC are higher than the levels in the literature that show a causal association between those chemicals and RCC. According to Bove 2014a's cumulative exposure charts, Mr. Howard would have been categorized as being in the "medium" exposure group for both Benzene and VC.⁷ These correspond to HR of 1.38 (Benzene) and 1.61 (VC).⁷

There is evidence to support the causal relationship of benzene and vinyl chloride to kidney cancer at least as likely as not and using an equipoise standard.

d. Conclusion For All Chemicals

There was a monotonic response relationship for TVOCs at Camp Lejeune and kidney cancer. Bove 2014a.⁷ The HRs for this analyses were 1.42 (low exposure), 1.44 (medium exposure) and 1.54 (high exposure).⁷

The epidemiological and occupational studies collectively underscore the link between TCE, PCE, benzene, and vinyl chloride exposures and renal cell carcinoma. Findings from Camp Lejeune studies, occupational analyses, and dose-response models reinforce the carcinogenic roles of these chemicals, particularly in populations with high or prolonged exposures. Mr. Howard's exposure meets the medium exposure groups from each of the four chemicals in the Bove 2014a study.⁷ These categories correspond to significant increased risks of kidney cancer, as described above.

Mr. Howard would meet and exceed other levels in the literature relating to the toxins at issue and their association with kidney cancer.

The data from the Camp Lejeune studies of Bove and ATSDR provide compelling data that exposures of this kind are causally related to kidney cancer.^{7,17}

11. Patient-Specific Considerations

Age and Tumor Size:

[REDACTED]

Latent Period:

[REDACTED].

Exposure: Mr. Howard's TCE exposure (5,937 µg/L) far exceeds safe thresholds and aligns with levels implicated in RCC cases. The reports of Drs. Hatten and Bird detail the levels at which the chemicals at issue have been known to be causally associated with kidney cancer. I have read the reports and agree with the findings in those reports as to the levels that are known to cause kidney cancer. For example, some of the levels known to cause kidney cancer, relating to these toxins, are as follows:

- **Cumulative exposure to 27.1-44.1 mg of PCE⁹**
- **Sustained exposure to 0-25.3 ppb of TCE¹⁸**